

PREVENTING SUDDEN CARDIAC DEATH IN YOUNG ATHLETES

by

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Preface

This literature review will serve as a summary of research that is pertinent to the study of sudden cardiac arrest (SCA) and sudden cardiac death (SCD) in young athletes at the high school, collegiate, and early professional level. For the purpose of this paper, a young athlete is considered to be an individual under the age of 30 that participates in regular, intense, and organized training with the purpose of performing well in competition (Casa & Stearns, 2017). It will outline the prevalence, various causes of SCA and SCD, prevention measures, pre-participation screenings, as well as distinguish the difference between SCD and SCA from a myocardial infarction. This review will also include case studies that were provided through interview and published articles.

Physiological changes to an athlete's heart

As an athlete becomes more conditioned, physiologic changes occur in the heart. These changes depend on the type and amount of exercise performed and in order to acquire these adaptations, physical activity must be regular and intense (Whyte, Loosemore, & Williams, 2016). These changes are typically minimal, non-pathologic and they allow an athlete to maximize cardiac output during exercise (Whyte et al, 2016). Cardiac output is the product of heart rate and stroke volume. During exercise, the cardiac output increases because of the increase in oxygen the muscles need to continue to perform (Whyte et al, 2016). This increase in cardiac output is completed through an increase of both heart rate and stroke volume, with heart rate being the main factor (Whyte et al, 2016). As conditioning continues, hypertrophy of the left ventricle occurs meaning there is an increase in ventricle size, heart wall thickness, and diastolic filling (Whyte et al, 2016). These adaptations all increase an individual's stroke volume. This increase in stroke volume increases cardiac output at rest and during exercise. Furthermore, due to this increase in stroke volume, a lower resting heart rate is observed in highly trained athlete's (Whyte et al, 2016).

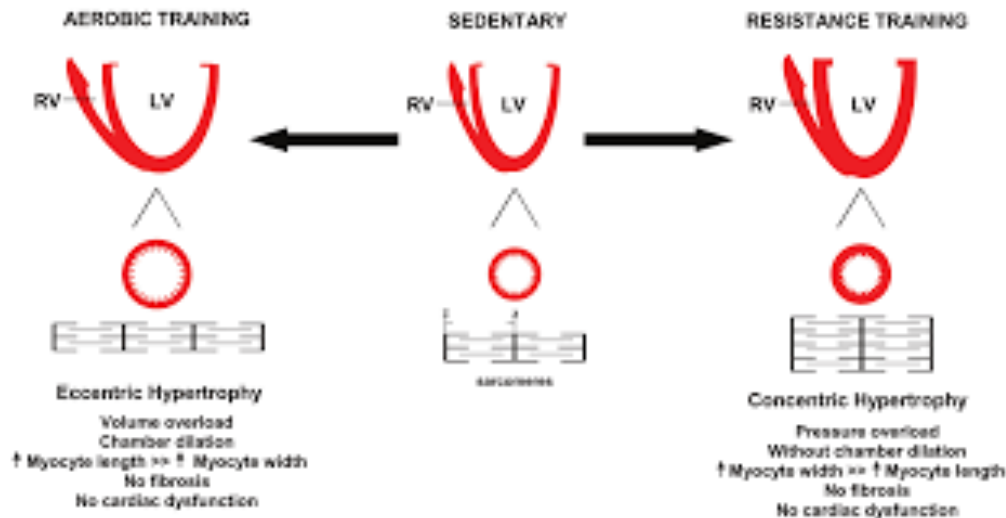


Figure 1: Eccentric and concentric cardiac hypertrophy induced by exercise training: microRNAs and molecular determinants

The difference in hemodynamic needs of different types of conditioning determines the type of cardiac modeling that will occur (Whyte et al, 2016). There are 2 main types of adaptation of the left ventricle; concentric and eccentric left ventricular hypertrophy (Fernandes, Soci, & Oliveira, 2011). These changes are shown in Figure 1 from the Brazilian Journal of Medical and Biological Research in an article discussion eccentric and concentric cardiac hypertrophy induced by exercise (Fernandes et al, 2011). Concentric is characterized by an increase in left ventricular wall thickness and little to no amount of chamber dilation. The increase in wall thickness is non-pathologic and is caused by a pressure overload often seen in heavily resistance trained athletes (Fernandes et al, 2011). Eccentric left ventricle hypertrophy is characterized by chamber dilation and little to no increase in ventricular wall thickness. This hypertrophy is also non-pathologic and is a result of volume overload (Fernandes et al, 2011). Sports that require athletes to be highly trained in both aerobic and resistance activities may see an overlap in hypertrophies, showing increased wall thickness and an increase in the left ventricle chamber size (Whyte et al, 2016).

Sudden cardiac arrest and sudden cardiac death

Sudden cardiac death (SCD) is defined as the sudden death of an individual due to cardiac-related causes within an hour of symptom onset (Katritsis, Gersh, & Camm, 2016). Sudden cardiac arrest (SCA) is when these cardiac-related issues cause a decrease and stopping of heart function but does not result in death (Katritsis et al, 2016; Drezner, Peterson, Seibert, Thomas, Lopez-Anderson, Suchsland, & Kucera, 2018). This differs from a heart attack in that a heart attack is a blockage in an artery that leads to a decrease in blood flow to heart muscle (Whyte et al, 2016). There are a variety of causes associated with SCA and SCD in young athletes that include structural, electrical, and inherited cardiac abnormalities (Oliva, Grassi, Campuazno, Brion, Arena, Partemi, & Brugada, 2016). Most of these causes have no symptoms or warning signs and often SCA is the first sign of any cardiovascular disorder (Drezner et al, 2018; Maron, Haas, Murphy, Ahluwalia, & Rutten-Ramis, 2014). It is hard to record SCD in young athletes because there is no mandatory reporting of SCD in athletes across all levels of athletics from high school to professional and there is a struggle in defining what characterizes a competitive athlete (Casa & Stearns, 2017; Luna, 2000). In athletes, the hemodynamic demand of vigorous activity triggers arrhythmias in hearts with underlying diseases (Oliva et al, 2016; Casa & Stearns, 2017). Among the common causes of SCA and SCD in young athletes, some of the most prevalent are hypertrophic cardiomyopathy (HCM), commotio cordis, myocarditis, aortic rupture in Marfan syndrome, and arrhythmogenic right ventricular cardiomyopathy (ARVC) (Maron et al, 2014). Because SCA and SCD are caused by an unknown underlying cardiovascular disorder, detection and prevention are extremely important. Detection of an underlying abnormality could be the difference between a young athlete living and dying.

Pathophysiology

There are many causes of SCD in young athletes that ranges from electrical to structural but ventricular fibrillation (VF) appears in approximately 40% of cardiac arrests that take place outside

of a hospital (Casa & Stearns, 2017). When initial cardiac rhythm is measured, the rate of VF in athletes that suffer from SCA is higher than 40% (Casa & Stearns, 2017). VF is a shockable rhythm and the use of an automated external defibrillator (AED) is successful in correcting it if used correctly and in a timely fashion (Casa & Stearns, 2017). Some of the top causes of SCA and SCD are listed and described in Table 1 but these are not all the causes and in some cases a diagnosis of SCA or SCD of idiopathic origin is given. This table is from the United States National Registry (Maron et al, 2016).

Sudden Death in Young Athletes: Demographics	
Autopsy-based survey of 387 United States Athletes	
<u>Etiology</u>	<u>Percent</u>
HCM	26.4%
Commotio cordis	19.9%
Coronary artery anomalies	13.7%
LVH indeterminate causation	7.5%
Myocarditis	5.2%
Ruptured aortic aneurysm (Marfan's)	3.1%
Arrhythmogenic RV cardiomyopathy	2.8%
Tunneled (bridged) coronary artery	2.8%
Aortic valve stenosis	2.6%
Atherosclerotic CAD	2.6%
Dilated cardiomyopathy	2.3%
Myxomatous mitral valve degeneration	2.3%
Asthma or pulmonary	2.1%
Heat stroke	1.6%
Drug abuse	1.0%
Other CV cause	1.0%
Long QT syndromes	0.8%

Table 1: Demographics of Sudden Cardiac Death Causes in the United States

Hypertrophic cardiomyopathy

In the United States, the most common cause of SCD is HCM (Thomas, 1996). The reported number of SCDs that occur due to HCM varies due to the lack of a mandatory reporting protocol. Studies have found it to account for anywhere from one third to one half of all SCD occurrences (Casa & Stearns, 2017; Thomas, 1996). HCM is a condition in which there is hypertrophy of the

left ventricle up to 16 mm in thickness or more (Casa & Stearns, 2017; Sheffle & Bowden, 2014). Normal left ventricle wall thickness is 12 mm or less and the ventricle is considered borderline if it falls between 13mm and 15 mm (Casa & Stearns, 2017). HCM impairs diastolic function of the ventricles, causes ventricles to become noncompliant, and leads to disorder of cardiac myocytes (Sheffle & Bowden, 2014). Contrary to concentric left ventricular hypertrophy, the enlarged ventricle wall can impair diastolic filling of the ventricle as well as impede blood flow out of the heart through the aortic semilunar valve in HCM (Casa & Stearns, 2017). HCM is inherited and an autosomal dominant abnormality that varies in expression. It occurs in about 1 out of every 500 adults and is usually asymptomatic (Patel & Elliot, 2012). Symptoms may present themselves as syncope, ventricular fibrillation, chest pain, as well as mimic symptoms of other CVDs (Patel & Elliot, 2012). Although this abnormality accounts for such a high number of deaths, it is often not diagnosed in current pre-participation screening protocols (Patel & Elliot, 2012).

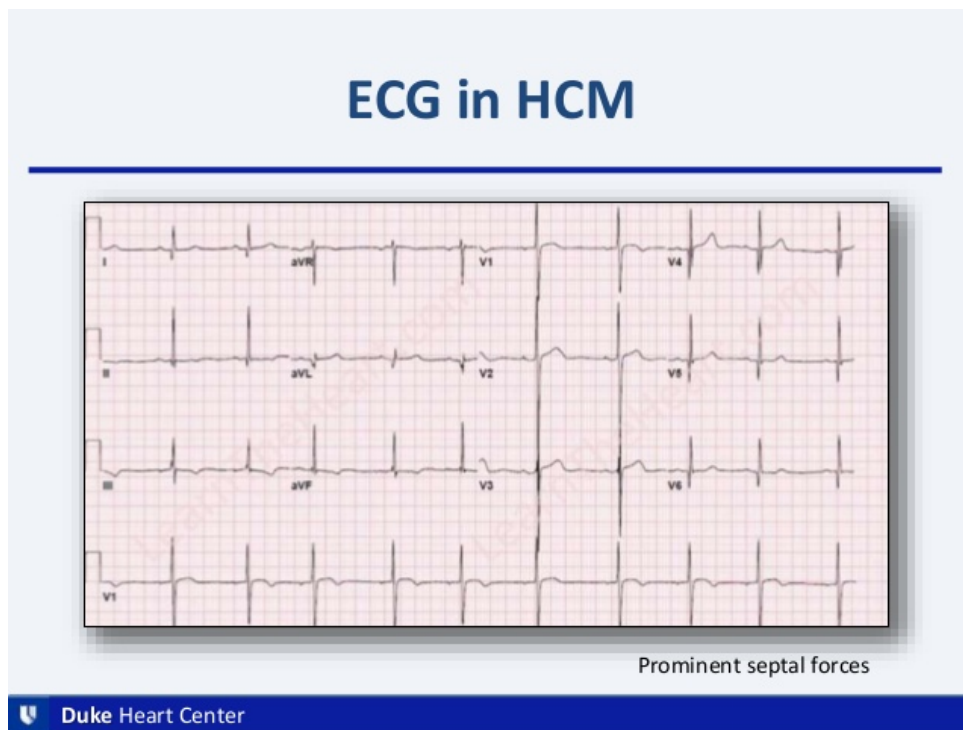


Figure 2: ECG of patient with HCM from Duke Heart Center

Athletes with HCM will have abnormal ECG results in 95% of cases (Casa & Stearns, 2017). HCM is hard to diagnose without an echocardiogram because on an ECG, characteristics of HCM and the physiologic change in an athlete's heart look similar (Corrado & Zorzi, 2018). Figure 2 shows an example of an ECG of a patient with HCM from the Duke Heart Center released in 2016 (Heart, 2016). For example, concentric changes to the heart as a result of resistance training cause the increase in thickness in the heart wall. HCM is also an increase in the thickness of the heart wall, although, it is pathologic and impedes on blood flow throughout the heart. Nevertheless, these increases in thickness, though different, cause similar outcomes on an ECG (Casa & Stearns, 2017). 4 to 6 weeks of deconditioning may be used to distinguish between HCM and athlete's heart when using an ECG but this deconditioning period may be detrimental to performance in a highly competitive athlete (Casa & Stearns, 2017).

Commotio cordis

Commotio cordis is one of the few causes of SCD and SCA that cannot be predicted. Commotio cordis is an arrhythmia or full interruption of heart rhythm that is a result of an impact to the chest area over the heart (Davey, Quintana, & Upadhyay, 2018). This impact often leads to VF which is lethal if not treated (Davey, Quintana, & Upadhyay, 2018). Commotio cordis is the second leading cause of SCD in young athletes and is unpredictable (Maron, 2012; Davey, Quintana, & Upadhyay, 2018). It can happen in a variety of sports including limited-contact sports by accident (Davey, Quintana, & Upadhyay, 2018).

Myocarditis

Myocarditis is characterized by inflammation of the myocardium (Casa & Stearns, 2017). It affects the heart muscle and electrical system leading to arrhythmias that cause SCA or SCD (Casa & Stearns, 2017; Myocarditis). Myocarditis is often caused by a viral infection but can also be the

result of a poor reaction with a drug (Myocarditis). Myocarditis can be diagnosed using ECG, a chest X-ray, MRI scans, and an echocardiogram among other tests, but it is also typically accompanied by signs and symptoms such as chest pains and shortness of breath that act as red flags (Myocarditis).

Marfan syndrome

The physical examination looks for physical signs of Marfan syndrome which most commonly refers to long limbs (Thomas, 1996). In most individuals, a person's height is greater than their wingspan but one of the most common signs of Marfan syndrome is the wingspan length exceeding the height of the individual (Thomas, 1996). Marfan syndrome is commonly associated with aortic dilation, aortic dysfunction, and an increased risk of aortic dissection (Thomas, 1996). Early detection of Marfan's syndrome is important because a ruptured aorta due to Marfan's accounts for 3.1% of all SCDs (Myerson, Sanchez-Ross, & Sherrid, 2012).

Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an abnormality in the right ventricular myocardium (ARVC). This abnormality causes the breakdown of the heart muscle which increases the chance of having a lethal arrhythmia (ARVC). Some of the symptoms associated with ARVC are light-headedness, fainting, and a feeling of pounding in the chest but these symptoms are often not present in the early stages (ARVC). ARVC can be caused by many different gene mutations and seems to be genetically linked. ARVC can be diagnosed using an ECG (Arrhythmogenic).

Athletic fatalities

Athletic fatalities are split into 2 categories; direct and indirect (Casa & Stearns, 2017). Direct refers to a death resulting directly participating in skills and drills associated with the sport (Casa & Stearns, 2017). Indirect refers to a systemic failure that occurs as a result of the exertion from participation in the sport (Casa & Stearns, 2017). SCD falls into the indirect category because it is

a failure of the cardiovascular system to perform due to the physical requirements of participating in the sport.

Prevalence

SCD is the leading cause of exercise-induced death for young, competitive athletes, accounting for approximately 75% of all deaths (Drezner et al, 2018). It is hard to know the exact frequency of SCD in the young athlete population because of there are no regulations on reporting and it isn't necessary to report a SCD occurrence at all (Drezner et al, 2018). Because the frequency of SCD in young athletes is anywhere from 1:9000 to 1:300000 depending on the study, there is a need for a mandatory reporting system to be in place in the United States (Casa & Stearns, 2017). A reporting system, along with a universal definition of the term athlete in this context, would acknowledge the definite prevalence of SCD across all levels of competitive sport, from high school to professional. This information would assist in assessing the current pre-participation screening and develop additions to these screening to, hopefully, decrease the frequency of SCA/D in young competitive athletes. The current definition of a competitive young athletes is an individual under 30 that competes in an organized team or individual sport that strives for excellence in mandatory competitions (Whyte et al, 2016). SCD is more common in males than females and most prevalent in U.S. athletes that were of African-American descent (Maron, Haas, Ahluwalia, Murphy, & Garberich, 2016). The highest incidence rate was in basketball, football, and track respectively (Whyte et al, 2016). SCA and SCD are actually seen at a higher frequency in the general population but because of the high health and fitness level of highly trained athletes, the occurrence of SCA and SCD is typically more unexpected and impactful.

Prescreening Process

In the United States, athletes at the high school and collegiate level are usually required to fill out a health history form and have a physical exam performed by a physician or athletic trainer yearly. Most professional sports teams also require a health history and physical exam that includes heart rate and blood pressure at the beginning of each season (Piper & Stainsby, 2013). Some teams include an electrocardiogram (Mosterd, 2018) in their pre-season physical but this is not common and not required (Riebe, Ehrman, Liguori & Magal, 2018; Piper & Stainsby, 2013). Pre-screening is the first line of defense in SCA and SCD stressing the importance of efficient and accurate testing.

American Heart Association pre-participation screening protocol

Table 2. American Heart Association Recommendations on Screening for Cardiovascular Abnormalities in Competitive Athletes
<p>Personal history</p> <ul style="list-style-type: none"> Elevated blood pressure Excessive dyspnea or fatigue associated with exercise Exertional chest pain or discomfort Prior recognition of a heart murmur Unexplained syncope or near-syncope <p>Family history</p> <ul style="list-style-type: none"> Disability from heart disease in a close relative younger than 50 years Premature death (sudden and unexpected, or otherwise) before 50 years of age due to heart disease Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long QT syndrome, Marfan syndrome, or arrhythmias <p>Physical examination</p> <ul style="list-style-type: none"> Brachial artery blood pressure (sitting position) Femoral pulses to exclude aortic coarctation Heart murmur* Physical stigmata of Marfan syndrome
<p><small>*—Auscultation should be performed in the supine and standing positions (or with the Valsalva maneuver) to identify murmurs of dynamic left ventricular outflow tract obstruction.</small></p> <p><small>Adapted with permission from Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. Circulation. 2007;115(12):1646.</small></p>

Table 2: 12-point Pre-Participation Screening for Athletes by AHA

Pre-participation screenings became an initiative in 1996 and was supported by the American Heart Association (AHA), but there are no current regulated guidelines (Subasic, 2009). In a policy

guidance released by the AHA, they revealed that only about 6% of doctors in Washington State followed the guidelines they set out (Preparticipation). There is a 12-point assessment that involves a physical examination and a health history that must be verified by a parent for minors (Subasic, 2009). This 12-point assessment is shown in Table 2 from the American Heart Association. The health history questionnaire targets the possibility of heart-related abnormalities based on personal and familial history (Subasic, 2009). The questionnaire contains 5 questions pertaining to chest pain, syncope, excessive fatigue in the presence of exercise, heart murmur and elevated blood pressure that may or may not be present in the athlete (Subasic, 2009). Following this questionnaire are 3 questions pertaining to history of sudden death, heart disease before age 50, and any other cardiovascular condition that may be present in the patient's family (Subasic, 2009). The four elements of the physical exam include physical appearance of Marfan syndrome, resting blood pressure from both arms while sitting, listening for presence of heart murmur, and palpation of the femoral pulse (Subasic, 2009). Those performing the physical, qualified practitioners, also listen for heart murmur when the patient moves from sitting to standing as well as while performing the Valsalva maneuver (Subasic, 2009). It is recommended that these pre-screenings take place annually and if any of the 12-points indicate a possible cardiac abnormality, the patient is recommended for further evaluation before being allowed to participate (Subasic, 2009). Professional sports in the United States, however, have adopted the European Society of Cardiology screening that includes a 12-lead ECG along with the health history and physical examination (Corrado, Basso, Schiavon, Pelliccia, &Thiene, 2008).

European Society of Cardiology pre-participation protocol

The European Society of Cardiology (ESC) has a similar protocol to the AHA when performing a pre-participation screening on athletes with one major difference; a 12-lead electrocardiogram (Mosterd, 2018).

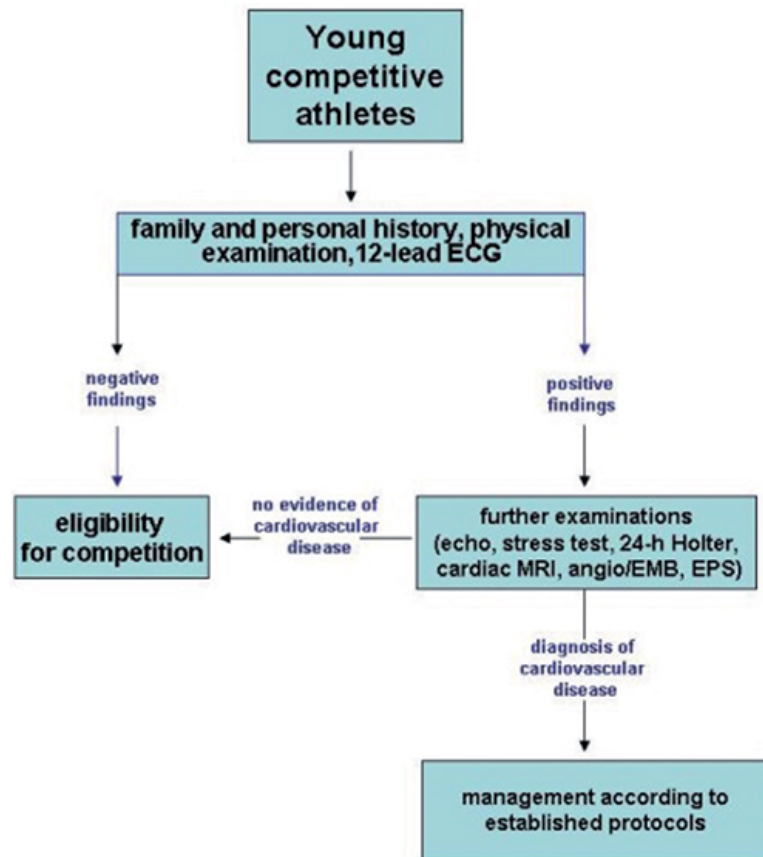


Figure 3: Sudden Cardiac Death in Young People and Athletes; flowchart of ESC pre-participation screening protocol

The overall pre-participation process is shown in Figure 3 from an article in the Italian Journal of Medicine (Corrado & Zorzi, 2018). Phase one of the screening is a family and personal health history, a physical examination, and a 12-lead ECG. If these all have negative findings, then the athlete is cleared for competition. If there are any concerns regarding one of these factors, the patient is referred for further examination that includes an echocardiogram, stress test, 24-hour Holter monitor, a magnetic resonance imaging (MRI) scan of the heart, and/or an angiography among any other test a cardiologist deems necessary (Corrado & Zorzi, 2018). If results come back negative, the athlete is free to participate, and if positive, the athlete starts treatment for underlying cardiovascular condition.

Ethical and Legal Considerations for Pre-Participation Screenings

For athletes that are associated with a school or college, the school system or NCAA is ethically responsible for producing a cost-effective and efficient pre-participation screening to maximize the safety of their athletes (Maron 2012). Although the ethical responsibility falls on the institutions where the athlete is participating at, there is no real legal responsibility on these places to provide an adequate screening (Maron 2012). This legal responsibility does not fall on the physician or any other medical professional that clears the athlete for participation in exercise either and to form a malpractice suit against the physician requires adequate proof which is hard to come by (Maron, 2012). Because there are no legal responsibilities to either the institution or medical professional, where does it fall? There aren't mandates on reporting SCA and SCD in young athlete's either, so how are athlete's being protected in the case that a faulty pre-participation screening doesn't catch a cardiac abnormality resulting in the athlete's death?

ECG addition to the AHA model

There is evidence that an ECG included in the pre-participation screening of young athletes can help identify cardiovascular conditions that a physical examination or health history can't (Casa & Stearns, 2017). Because of costs and access, the AHA decided not to include ECG in the screening in 2007 (Subasic, 2009). Adding the ECG has been a topic of debate for years due to the possibility of false-positive results and lack of resources (Subasic, 2009). The concern in including ECG in the screening process revolves around the possibility of false-negative as well as false-positive results. One study on the Italian model of screening found an overall 7% false positive rate and an overall 2% disqualification rate (Casa & Stearns, 2017). Because the number of individuals that participate in athletics in the United States is much greater than that of Italy and assuming the percentages stayed the same country to country, the amount of false-positives and therefore disqualification from sport would be undesirable (Casa & Stearns, 2017). But do the

benefits outweigh the risks of not using ECG? One study showed that 95% of patients that suffered from SCD had conditions that could have been found using an ECG (Casa & Stearns, 2017). A single ECG prior to the beginning of athletics in high school may be beneficial to the athlete's career and more cost-efficient than a repeated scan every year. ECG can pick up a multitude of abnormalities including HCM which is the number one cause of SCD in athletes in the United States (Casa & Stearns, 2017). HCM can present itself as early as early childhood and as late as mid adulthood and is often asymptomatic (Casa & Stearns, 2017). Because it can develop between any of these ages, a single ECG, while cost effective, may not be adequate to identify those individuals that have the genetic disorder. The main question to be asked, does the possibility of false-positives and false-negatives outweigh the cases that could be caught and prevented?

Emergency response to SCA

Because the pre-participation screening can't predict when, where, and to whom a SCA will happen, emergency preparedness is extremely important. Emergency response, meaning cardio pulmonary resuscitation (CPR) and automated external defibrillator (AED) use, is considered secondary prevention methods for SCD (Casa & Stearns, 2017). For young athletes, this means quick and easy access to an AED, an athletic trainer on site, and an emergency preparedness plan in place are all vital to SCA outcome.

AED access

An AED should also always be available to coaches, athletes, athletic trainers, or anyone else that is present at an athletic event. The AED is crucial in the survival of individuals who suffer from SCA. Survival of a cardiac arrest increases from 9% with CPR only, to 38% with the use of CPR in conjunction with an AED (Piper & Stainsby, 2013). CPR and AED are considered secondary management for SCA cases and pre-participation screenings are considered the primary protection stressing the importance of an adequate screening protocol. Because VF

occurs in 40% of SCA cases, and it is a shockable rhythm, AED access is essential to survival of these athletes (Casa & Stearns, 2017). While there are some mandates put on AED placement, not all individuals present at a practice or competition know where it is located. It should be a part of the first team meeting and should be clearly visible and accessible to anyone present. AED legislation differs state to state and in North Carolina there are currently no laws regarding AED maintenance or notification of use (North Carolina State AED law).

CPR certified individuals

Although it has been seen that AED use has a higher percentage of survival, CPR use is also important to save lives. CPR should be started immediately and used while the AED is being retrieved. For this reason, it is vital to have individuals that are CPR certified present at every team practice, conditioning day, and competition (Drezner et al, 2018).

Mandatory Reporting

There is no mandatory reporting in the case of a SCA or SCD (Casa & Stearns, 2017). While the occurrence does often catch the attention of the media, it can just as easily be swept under the rug. A mandatory reporting system would allow for easier collection on the data surrounding sudden cardiac events and give even further insight into the most common causes and further improvement to prevention measures.

Case Studies

When an athlete suffers from a sudden cardiac arrest and survives, it gives professionals an insight into what might be happening structurally, electrically, or otherwise. The following two case studies show how unpredictable SCAs can occur and how even advanced medical screenings may not help predict these occurrences. Omar Carter's story was provided via personal interview and the case of the hockey player was a case study published in an academic journal. Both stories stress the importance of emergency preparedness and immediate response.

Hockey player

This case study comes from a 23-year-old hockey player that suffered an SCA while playing in a hockey game. There were no previous symptoms, the player had not recently taken any contact, and there was no history of trauma (Piper & Stainsby, 2013). It took EMS 12 minutes to get there from the start of CPR and other players used an AED to restart his heart (Piper & Stainsby, 2013). He was transported to the hospital and an initial ECG was taken (Piper & Stainsby, 2013). It showed a decrease in left ventricular function (Piper & Stainsby, 2013). The second ECG taken later showed no dysfunction but a dilated right ventricle attributed to his status of an athlete (Piper & Stainsby, 2013). He had multiple tests taken which showed normal coronary arteries, no evidence of a myocardial infarction, no HCM, no scarring, and no fibrosis so he was diagnosed with a SCA of idiopathic origin (Piper & Stainsby, 2013). He received an implantable cardioverter-defibrillator (ICD) and still has not been cleared to return to play (Piper & Stainsby, 2013). All tests following discharge from the hospital came back with no abnormalities and the cause is still unknown (Piper & Stainsby, 2013). As a part of pre-participation screenings for his team, he had been given a yearly physical that included checking heart rate and blood pressure as well as an ECG prior to the SCA (Piper & Stainsby, 2013). There had been no abnormalities reported in these previous tests either (Piper & Stainsby, 2013). A secondary method of prevention in this situation saved this individual's life, demonstrating the importance of the use of CPR and an EAD.

Omar Carter

Omar Carter was an athlete his whole life. He played a multitude of sports starting at a young age but it was pain in his arm while he was doing push-ups when he was 16 that initially brought him into the doctor. He described pain in his left arm during conditioning and was taken to the hospital as a precaution. An ECG and an echocardiogram were performed on him there and his heart was considered borderline for HCM. There was no family history of any heart condition and even his twin brother did not exhibit these same abnormalities. His first doctor made him stop all sports

until there were more tests run and ultimately Carter changed doctors to complete these tests. His second diagnosis was an unknown arrhythmia. Following this diagnosis, Carter was permitted to rejoin his sports teams but had to have tests including ECG, echocardiogram, stress test, and wearing a portable heart monitor every 3 months for the next year. The next year the tests moved to every 6 months and after that the tests were performed annually. As he transitioned into collegiate athletics on the Appalachian State Men's Basketball team, he continued these annual exams that aligned with a more extensive NCAA athletic physical. He was only allowed to play basketball and wasn't allowed to perform any exercise that was considered too strenuous. He made it through college without any incidents and moved on to play professional basketball in the Dominican Republic. It was during warm-ups of one of his professional games that Carter collapsed on the court and his heart stopped for approximately 12 minutes. He credits a nurse in the crowd with saving his life due to her quick action of immediately starting CPR. He was revived with an AED and spent the next couple of months in the hospital. While there he was given 3 shots a day (one in the stomach and 2 in the arm) to treat a low potassium level. After his hospital stay, Carter's diagnosis was changed to "undiagnosed high blood pressure" and this remains his current diagnosis. A portable defibrillator was the first treatment and then in 2013 he had an ICD placed. He still has memory loss surrounding his cardiac arrest and has not played basketball or any sport since. Exercise that is permitted for him are light biking or walking and he maintains a heart-healthy and balanced diet. When asked what he would change about current pre-screening processes, he suggested adding testing such as an ECG and/or stress test to the mandatory pre-participation guidelines. He also pushed for more extensive participation in programs like Heart of a Champion. Carter is an example of an athlete that had all the tests and saw the cardiologist regularly but still had a SCA. His story stresses the importance of immediate emergency

preparedness and response. Carter has since started the Omar Carter Foundation where he teaches people bystander CPR (O. Carter, personal communication, February 23rd, 2019).

Conclusion

While it may be impossible to diagnose and prevent all cases of SCA and SCD in young athletes, including an ECG in the pre-participation screening can increase the current number of incidences. Mandates should also be implemented on mandatory reporting, emergency action, and AED placement.

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